undergoing coronary angioplasty. This study was designed to determine whether an optimized weight-adjusted heparin regimen was more effective in suppressing procoagulant activity during coronary interventions.

Methods: In 94 patients undergoing coronary revascularization a bolus of 100 U/kg of heparin was administered followed by an infusion of 20 U/kg/hr. Fibrinopeptide A (FPA) and prothrombin fragment (F1.2), markers of thrombin and factor Xa activity, respectively, were measured in coronary sinus blood samples collected with heparin-bonded catheters.

Results: The activated coagulation time (ACT) was 347 ± 41 seconds and the plasma heparin level was 2.75 ± 0.13 U/mL. During interventions mean concentrations of FPA decreased from 3.9 ± 0.4 nmol/L at baseline to 2.2 ± 0.3 nmol/L at the conclusion of the procedure ($P = 0.0001$). However, persistent increases in FPA (>3.0 nmol/L) were detected in up to 30% of patients despite maintaining ACT values >300 seconds by the Hemochron method. FPA concentrations were increased in patients with significant coronary dissection (4.6 ± 0.8 versus 2.5 ± 0.2 nmol/L, $P = 0.002$) and threatened or abrupt coronary occlusion (5.6 ± 1.2 versus 2.7 ± 0.2 nmol/L, $P = 0.001$). F1.2 concentrations were not different.

Conclusions: Thus, weight-adjusted heparin dosing resulted in marked increases in heparin levels and the ACT. However, despite optimized heparin dosing, thrombin activity persists in up to 30% of patients and is associated with significant coronary dissections and the development of in-lab threatened or abrupt coronary occlusion.

4:15

911-2 Ticlopidine Reduces Plasma Tissue Factor Levels in Unstable Patients Undergoing Dilation Procedures

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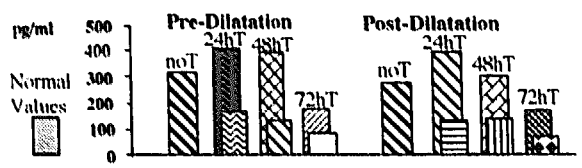
Coronary Stenting (S) ablate and fractures the endothelium, exposing tissue factor (TF) to factor VII. Plasma TF levels collected in the coronary artery might be an index of plaque TF expression. Aim of the study was to investigate if the combination of 250 mg/d ASA pretreatment and 250 mg/bid ticlopidine (T) given for 24, 48, or 72 h may influence TF levels in stable (SA) versus unstable angina (UA) pts. We selected 100 patients (pts) undergoing S for a 77 ± 3% stenosis. Pts received 250 mg/d ASA and ticlopidine (T). They were considered for 24 h vs 48 h or >72 h T and for SA or UA. TF was measured before 150 U/Kg heparin (H) injection and before dilation from the coronary ostium. Measurements were repeated after S. Results shown in Figure indicate that 72 h T, ASA and H reduce TF expression at levels observed in normal subjects both in stable and in unstable angina despite significant differences between SA (smal bars) and UA.



noT: no-Ticlopidine = SA patients treated with ASA and PTCA

Conclusions: Ticlopidine also decreases coagulation activation by reducing TF expression.

4:30

911-3 Impact of Cilostazol on Neointimal Proliferation Following Palmaz-Schatz Stent Implantation: A Prospective Randomized Trial

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We previously reported that cilostazol, a new synthetic platelet-aggregation inhibitor, inhibits intimal proliferation following directional coronary atherectomy and that it also significantly reduces restenosis following balloon angioplasty. The aim of this study was to determine the effect of cilostazol on neointimal proliferation following Palmaz-Schatz (P-S) stent implantation. Twenty nine lesions in which P-S stents were successfully implanted were randomly assigned to a cilostazol (C) (200 mg/day) group (15 lesions, 18 stents) or an aspirin (A) (250 mg/day) group (14 lesions, 23 stents). Medication was started just after procedure and was continued to six month follow-up in all patients. IVUS was performed at post procedure and follow-up (Fu). Mean Fu duration was 194 ± 64 days. Stent area (SA) and lumen area (LA) were measured serially at the edge and the body of P-S stents. Neointima area (NA) was calculated as SA - LA and Fu %NA was calculated as Fu NA/Fu SA × 100. IVUS measurement were performed blindly.

	post SA (mm^2)	Fu SA (mm^2)	Fu NA (mm^2)	Fu % NA (%)
C group edge (n = 15)	7.02	7.40	1.58	22.15*
A group edge (n = 14)	6.54	6.90	2.15	32.16
C group body (n = 15)	7.05	7.58	1.58*	21.85*
A group body (n = 14)	6.61	7.05	2.17	31.81

*p < 0.05 vs A group

Conclusion: Cilostazol has an inhibitory effect on neointimal proliferation following P-S stent implantation.

4:45

911-4 Pravastatin Prevents Ischemic Events After Angioplasty in Myocardial Infarction Patients With an Average Cholesterol

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Background: Hmg CoA reductase inhibitors result in modest angiographic regression in patients with coronary disease and do not prevent restenosis in patients after PTCA. Do they prevent clinical events in post PTCA patients?

Methods: In the Cholesterol and Recurrent Events (CARE) trial, patients with a proven myocardial infarction and an average cholesterol level were found to have reduced ischemic events with pravastatin. All had serum cholesterol values <240 mg/dl. In this study 1369 patients had PTCA between an index myocardial infarction and randomization. PTCA patients received either pravastatin (n = 701) or placebo (n = 668) and were similar in baseline characteristics including cardiac risk factors. After up to 5 years of follow-up, patients on pravastatin were compared with placebo.

Results:

	Relative Risk (CI)	p value
1) Coronary disease death	0.91 (0.52–1.59)	0.728
2) Myocardial infarction	0.58 (0.40–0.86)	0.007
3) Bypass surgery	0.76 (0.52–1.09)	0.132
4) Repeat PTCA	0.84 (0.63–1.11)	0.222
5) Any revascularization	0.78 (0.31–0.98)	0.034
6) Combined (1 + 2 + 3)	0.75 (0.61–0.93)	0.009

Conclusion: Pravastatin prevents ischemic events and revascularization procedures in myocardial infarction patients with average cholesterol values after angioplasty.

912 Doppler Assessment of the Significance of Coronary Stenoses

Wednesday, April 1, 1998, 4:00 p.m.–5:00 p.m.
Georgia World Congress Center, Room 360W

4:00

912-1 Additive Value of Relative Coronary Flow Reserve in the Assessment of Intermediate Coronary Artery Stenosis: Comparison to Myocardial Stress Perfusion Imaging

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Absolute coronary vasodilator reserve (aCVR) is influenced by microcirculatory and hemodynamic perturbations. Relative CVR (rCVR = $aCVR_{target}/aCVR_{reference}$) should nullify these confounding conditions. Thus, we examined 50 pts at cath with thallium or sestamibi nuclear perfusion stress tests. In 22 pts with positive stress test, both aCVR and rCVR were lower than in 28 pts with negative tests (1.7 0.5 vs 2.56 ± 0.6; p < 0.0001 and 0.71 ± 0.2 vs 0.93 ± 0.15; p < 0.0002, respectively). For previously defined abnormal cutoff values for aCVR (<2.0) and rCVR (<0.8), rCVR had lower sensitivity than aCVR but with equal specificity (see table).

	Neg test	Pos test	p value	Sens	Spec
aCVR	2.56 ± 0.6	1.70 ± 0.52	<0.0001	77%	86%
rCVR	0.93 ± 0.15	0.71 ± 0.22	<0.0002	68%	86%

Concordant values of rCVR and aCVR significantly increased sensitivity (81%) and specificity (95%). When reference aCVR was abnormal, rCVR was abnormal in 2 pts with normal angiogram and 3 pts with normal stress tests. Abnormal rCVR was highly correlated to positive stress test (14/15) and angiographically significant stenosis (14/14). In pts with abnormal target aCVR, adding rCVR increases accuracy of the assessment of intermediate stenosis and should be part of direct physiological testing.